

# Guidelines for Insecticide Resistance Management (IRM) for foliar, seed and soil-applied insecticides



- ✓ Use effective cultural and biological control practices, including crop rotation, pest resistant or tolerant hybrids/varieties, and beneficial insect habitat enhancement and attractant methods.
- ✓ Scout fields on a regular basis. Before deciding to apply an insecticide, verify appropriate insect pest economic thresholds.
- ✓ When possible, select insecticides that conserve beneficial insects.
- ✓ Use insecticides at their recommended, labeled rates. Reduced (sub-lethal) doses select populations with tolerance to insecticides, while doses that are too high may impose excessive selection pressure for insect resistance.
- ✓ Follow the label and consult local extension service recommendations for advice on rotating insecticide classes as part of an IRM strategy.
- ✓ Monitor fields for insect pest resistance and assess control levels.
- ✓ If planting Bt corn hybrids, a refuge is required by law as part of an IRM program specific to Bt crops. Refuge corn acres do not contain the Bt insect trait used in the Bt planting. The refuge must be planted to 20% of the corn acreage on each farm, and there are specific configuration and distance requirements depending on Bt corn hybrid. Please consult UW Extension publication A3857 *Insect Resistance Management and Refuge Requirements for Bt Corn* for more information.

## ✓ See other side for more information on **How Insecticides Work**

\* Insecticide Resistance Action Committee

\*\*May be registered on crops other than corn (C), soybean (S) or wheat (W). See label for information.

# FOLIAR, SOIL AND SEED-APPLIED INSECTICIDES REGISTERED IN WISCONSIN FOR USE ON CORN, SOYBEAN, AND WHEAT

	SITE OF ACTION	IRAC* GROUP	CHEMICAL CLASS	ACTIVE INGREDIENT	PRODUCT EXAMPLES	REGISTERED ON**	COMPANY
<b>Insect Nerve Cell Action</b>	<b>Acetylcholine esterase inhibitors</b>	1	Carbamates	carbaryl	Sevin® XLR Plus	CSW	Bayer CropScience
				carbofuran	Furadan® 4F	CSW	FMC
				methomyl	Lannate® LV	CSW	DuPont
			Organophosphates	acephate	Orthene® 90S	S	Valent
				chlorpyrifos	Lorsban® Advanced or 15G	CSW	Dow AgroSciences
				dimethoate	Dimethoate® 4E	CSW	Chemnova
				malathion	Malathion® 5E	CW	Arysta LifeScience
				methyl parathion	Penncap®-M	CSW	United Phosphorus
				phorate	Thimet® 20G	CS	Amvac
	terbufos	Counter® 15G	C	Amvac			
	<b>GABA-gated chloride channel antagonists</b>	2	Phenylpyrazoles	fipronil	Regent® 4SC	C	BASF
	<b>Sodium channel modulators</b>	3	Pyrethroids	beta-cyfluthrin	Baythroid® XL	CSW	Bayer CropScience
				bifenthrin	Brigade® 2 EC	CS	FMC
				lambda-cyhalothrin	Warrior® II	CSW	Syngenta
				gamma-cyhalothrin	Proaxis®	CSW	Tenkoz
zeta-cypermethrin				Mustang Max®	CSW	FMC	
deltamethrin				Delta Gold® 1.5 EC	CS	Agrilience	
esfenvalerate				Asana® XL	CS	DuPont	
permethrin				Pounce® 3.2 EC	CS	FMC	
tefluthrin				Force® 3G	C	Syngenta	
<b>Nicotinic acetylcholine receptor agonists</b>	4	Neonicotinoids	clothianidin	Poncho®	C	Bayer CropScience	
			imidacloprid	Gaucho®	CSW	Bayer CropScience	
			thiamethoxam	Cruiser®	CSW	Syngenta	
<b>Nicotinic acetylcholine receptor allosteric activators</b>	5	Spinosyns	spinosad	Entrust®	CS	Dow AgroSciences	
<b>Premixes</b>	1+3	Organophosphate Pyrethroid	tebupirimphos cyfluthrin	Aztec® 2.1G	C	Bayer CropScience	
	1+3	Organophosphate Pyrethroid	chlorpyrifos gamma-cyhalothrin	Cobalt®	CS	Dow AgroSciences	
	3+3	Pyrethroid Pyrethroid	zeta-cypermethrin bifenthrin	Hero®	SW	FMC	
	3+4	Pyrethroid Neonicotinoid	cyfluthrin imidacloprid	Leverage® 2.7S	S	Bayer CropScience	
	3+4	Pyrethroid Neonicotinoid	lambda-cyhalothrin thiamethoxam	Endigo®	S	Syngenta	
	<b>Microbial disrupters of insect midgut membranes</b>	11B2	Bt subsp. <i>Kurstaki</i>	<i>Bacillus thuringiensis</i> subsp. <i>Kurstaki</i>	Dipel® DF	CS	Valent
<b>Inhibitors of chitin synthesis</b>	15	Benzoylureas	diflubenzuron	Dimilin® 25W	S	Chemtura	
<b>Ecdysone receptor agonists/molting disruptors</b>	18A	Diacylhydrazines	methoxyfenozide	Intrepid® 2F	C	Dow AgroSciences	
	18B	Azadirachtin	azadirachtin	Aza-Direct®	CSW	Gowan	

# How Insecticides Work

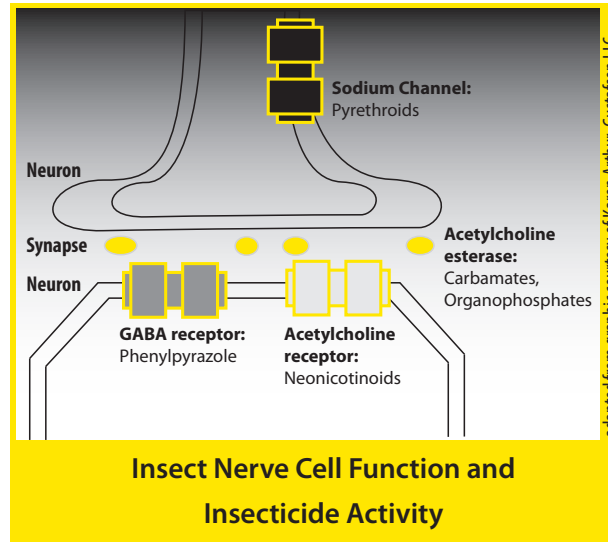
## Insecticide Resistance Definition

Resistance may be defined as 'a heritable change in the sensitivity of a pest population that is reflected in the repeated failure of a product to achieve the expected level of control when used according to the label recommendation for that pest species'. Cross-resistance occurs when resistance to one insecticide confers resistance to another insecticide, even when the insect has not been exposed to the latter product. Because pest insect populations breed quickly, there is always a risk that insecticide resistance may evolve, especially when insecticides are misused or over-used.

## Insect Nerve Cell Function

A majority of insecticides act on the insect nervous system. In order to understand insecticide modes of action, it's important to understand how nerve cells function. Basically nerve cells are a communication system that receives and transmits signals through the insect body. Incoming signals are transformed into electrical charges by specialized cells called neurons. Electrical charges move down channels in the nerve membrane by means of charged particles (ions). There are four types of channels to allow different ions to move along the neuron: sodium channels, potassium channels, calcium channels, and chloride channels. Many of the channels have gates that open or close in response to a certain stimulus.

Neurons transmit signals to other neurons and muscle fibers through a gap, called a synapse. When an electrical charge reaches the end of one neuron, it moves across the synapse to the receptor neuron with the aid of chemicals called neurotransmitters. Neurotransmitters transform an electrical signal into a chemical signal. The most common neurotransmitter is acetylcholine.



The neurotransmitter crosses the synapse and binds to a receptor on the receiving end of the next neuron, causing the chemical signal to be converted back into an electrical signal in the receptor neuron. Immediately after transmitting the signal across the synapse, the neurotransmitter is usually destroyed by enzymes, clearing the synapse. The transmitting nerve cell is then in a resting stage until the next signal is received. This process repeats over and over until the signal has reached the insect nerve bundles to be interpreted.

## Acetylcholine Esterase Inhibitors

Organophosphate and carbamate insecticides interfere with the transmission of nerve signals at the synapse. Organophosphate and carbamate insecticides function by inhibiting the action of the enzyme acetylcholinesterase. This causes a build up of acetylcholine in the synapse between neurons, resulting in the overstimulation of the nervous system.

## GABA-gated chloride channel antagonists

Phenylpyrazole insecticides such as fipronil affect the GABA receptor. GABA is a neurotransmitter

that when activated stops the nerve signal. When a phenylpyrazole insecticide binds to the GABA molecule, the neurotransmitter can no longer stop the nerve signal resulting in the overstimulation of the nervous system.

## Sodium Channel Modulators

Permethrin, lambda-cyhalothrin and esfenvalerate are all examples of pyrethroids. They prevent sodium channels from closing, resulting in continual nerve signal transmission resulting in the overstimulation of the nervous system.

## Nicotinic Acetylcholine Receptor Stimulation

Neonicotinoid insecticides mimic the action of the neurotransmitter, acetylcholine. Although acetylcholinesterase is not affected by these insecticides, the nerve is continually stimulated by the neonicotinoid, and the end result is similar to that caused by acetylcholinesterase inhibitors – overstimulation of the nervous system. Spinosad is also an acetylcholine receptor agonist. The exact mechanism of spinosad is somewhat different than that of the neonicotinoid class, but the end result is the same.

## Microbial disrupters of insect midgut membranes

*Bacillus thuringiensis* (Bt) is a bacterium that produces spores which contain a protein crystal with a toxic effect against some insects. When Bt is eaten by susceptible larva, it ruptures the lining of the insect's midgut and causes it to stop feeding and ultimately to die. Different strains of Bt produce slightly different crystals which have selective toxicity against various insects.

## Chitin Synthesis Inhibitors (CSIs)

Chitin is an important component of the insect's cuticle. Some insecticides, called chitin synthesis inhibitors, block the production of chitin. An insect affected by a CSI cannot make chitin and so cannot molt. Because molting must take place for

the insect to reach the adult stage, a CSI affected insect cannot reproduce. Eventually, the insect dies.

## Ecdysone receptor agonists/molting disrupters

Insects must molt, or shed their skin. Molting is necessary not only for the insect to grow through multiple life stages, but also to complete development to the adult stage so that it can reproduce. Disruption of, or interference with, molting hormones inactivates the molting process. Some insecticides target the insect's growth and development processes through interfering with hormones, and others through blocking the production of a structural component of the exoskeleton. This results in disruption of insect molting and preventing normal development, changes that cause the insects to die.

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